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Point-of-care ultrasound diagnosis of acute valvular emergencies

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ABSTRACT

Introduction: Acute valvular emergencies, a time-sensitive diagnosis, are nearly impossible to diagnose without ultrasound, and missing the diagnosis can significantly impact patient outcomes. Many emergency physicians lack access to echo technicians and may be uncomfortable performing the ultrasound themselves. Approaching the paucity of review articles, none of which are focused for the emergency physician, can be quite daunting, even for those with extensive ultrasound training.
Objective: Our goal is to provide a simple resource for emergency physicians to utilize ultrasound to diagnose acute valvular emergencies. We created a concise review that includes relevance and approach to the diagnosis, complete with live ultrasound images, reference cards, and focused figures for in-the-moment reference.
Methods: A team of ultrasound fellowship-trained emergency medicine physicians determined the highest yield emergent conditions and oversaw the project. Each team member reviewed the etiology, incidence, and level of evidentiary support for each condition and then provided a guide to the appropriate procedure and evaluation with relevant additional guidance. An in-house graphics team was consulted to prepare original, concise figures for easy reference. All sections of the manuscript and figures were reviewed for accuracy and ease of use.
Discussion: Critical aortic stenosis, infective endocarditis, left ventricular outflow tract obstruction, mitral stenosis, and mitral regurgitation were identified diagnoses for which ultrasound can provide immediate diagnostic information for emergency physicians.
Conclusion: The use of ultrasound in the emergency department setting is a critical adjunct to care for patients with acute valvular emergencies. One should always consider a comprehensive, cardiology-performed echocardiogram in these scenarios.
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1. Introduction

Acute valvular emergencies, time-sensitive diagnoses, are nearly impossible to diagnose without the use of ultrasound, and missing the diagnosis can significantly impact patient outcomes. Many emergency physicians (EPs) do not have feasible access to cardiac sonographers, nor do they feel comfortable performing the ultrasound themselves. Approaching the paucity of review articles, none focused on the EP, can be quite daunting, even for those with extensive ultrasound training.

Regardless, the importance of point-of-care ultrasound (POCUS) in the acute setting cannot be overstated, as it may drastically change management and save lives for patients with critical diagnoses. Further, POCUS and its scope have markedly evolved over the last two decades [1-3]. The focused questions of cardiac ultrasound (pericardial effusion/tamponade, systolic function, right heart strain) remain paramount. As EP's familiarity with POCUS expands, its utility has expanded to address additional, more advanced questions. These questions have allowed EPs to make critical diagnoses rapidly and, in many cases, have led to significant modifications in patient management [4]. We aim to provide a practical approach to incorporating cardiac POCUS into practice by providing guidelines for high-yield valvular emergencies. For each condition, we provide a basic description of the condition, the evidence related to POCUS-based diagnosis, how to perform a POCUS-based assessment and its evaluation, additional guidance, and associated pitfalls. In addition to the text, we have provided illustrated figures and quick guides to diagnosis, which provide concise, summative information to guide the EP. With this, we aim to help EPs work towards the most accurate diagnosis, best management, and

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appropriate disposition. Rapid diagnosis will allow the EP to make prompt management decisions that may involve referral, urgent comprehensive echocardiogram, or transfer, which are critical to the patient's immediate and long-term outcomes.

2. Methods

The authors used emergency ultrasound expert consensus to determine some of the most common and most clinically impactful acute valvular emergencies seen in emergency departments and searched PubMed and Google Scholar for related terms. This included clinical guidelines, retrospective and prospective (including clinical trials) studies, meta-analyses, and review articles. For this review, each valvular emergency was reviewed for prevalence, etiology, presentation, level of evidence for POCUS diagnosis, procedures for diagnosis with POCUS, and pearls and pitfalls from experts in cardiac ultrasound. To create the most approachable review, we made diagnosis reference cards and concise figures to serve as an in-the-moment guide to cardiac POCUS in patient diagnosis and management.

3. Discussion

3.1. Severe aortic stenosis

3.1.1. Description

Severe aortic stenosis (AS) is the most common valvular disease in the developed world, carrying an annual mortality rate of 25 % in symptomatic patients. The most common symptoms of AS are angina, syncope, and heart failure [5,6]. The most common causes of AS are congenital, calcific, and rheumatic diseases. Given the hemodynamics and preload-dependence of AS, POCUS is an essential tool for managing patients in undifferentiated shock [7].

The normal orifice area of the aortic valve (AV) is approximately 3 cm^2. The transvalvular pressure gradient increases significantly when the area is reduced by half, leading to left ventricular hypertrophy (LVH). LVH leads to impaired coronary blood flow and worsening heart failure. Consequently, increased oxygen demand causes angina [5]. Overall, the pathophysiology of AS leads to a preload-dependent lesion [6]. Echocardiography is the gold standard for diagnosing AS. Classically, a crescendo-decrescendo systolic ejection murmur is heard upon auscultation of the right upper sternal border; however, patients with critical disease, such as those presenting with syncope and shock, frequently do not have a murmur due to the reduction of stroke volume [5,6].

3.1.2. Evidence

Severe AS is a time-sensitive diagnosis made by echocardiography [5,6,8]. POCUS is useful in a wide range of cardiac cases in the ED, and POCUS-based AS assessment has been shown to be useful and quick in a variety of clinical settings [9-11]. The American College of Emergency Physicians (ACEP) endorsed emergency department (ED) echocardiography in 2001 and recommended that point-of-care ultrasound (POCUS) always be available. While not a replacement for comprehensive echocardiography, POCUS provides valuable information for rapid diagnosis and clinical decisions in the ED [12]. Emergency medicine (EM) physicians were asked if POCUS would have prevented or reduced adverse outcomes, and approximately 45 % reported that it would have drastically changed case outcomes, especially in cardiac cases [13]. In cases where immediate formal echocardiography is not feasible, POCUS is essential and highly accurate, even for novice users [5].

Specific to AS, there is a good inter-operator agreement between experts and novice users regarding qualitative and quantitative assessments of AS, especially severe AS [7,11,14-17]. There is a significant record that POCUS-based AS diagnosis is relatively quick and has high accuracy [11,14,15,18].

3.1.3. Assessment: procedure and evaluation

Severe AS is diagnosed by POCUS using qualitative and quantitative methods. The qualitative assessment utilizes parasternal long axis (PLAX) and parasternal short axis (PSAX) approaches to evaluate AV calcification, the presence of LVH, and AV leaflet mobility (Fig. 1a) [16]. One can visually appreciate increased echogenicity of the valves correlating with calcifications; usually, acoustic shadowing is seen once severe. Valvular thickening (mild, moderate, or severe) is assessed by evaluating the aortic cusps (Fig. 1b) [17]. An assessment of each cusp's mobility is done to determine the degree of restriction (none, mild, moderate, or severe). Qualitative severity scores assessing cusp restriction exist; however, they were omitted as they do not discern between moderate and severe disease and are less accurate [8].

The quantitative assessment involves three main physiologic parameters: AV_{area} via the continuity equation, peak aortic velocity (V_{max}), and mean transaortic gradient. AV_{area} is the area of the opening of the AV during systole and is normally >1.5 cm^2. Peak aortic velocity is the

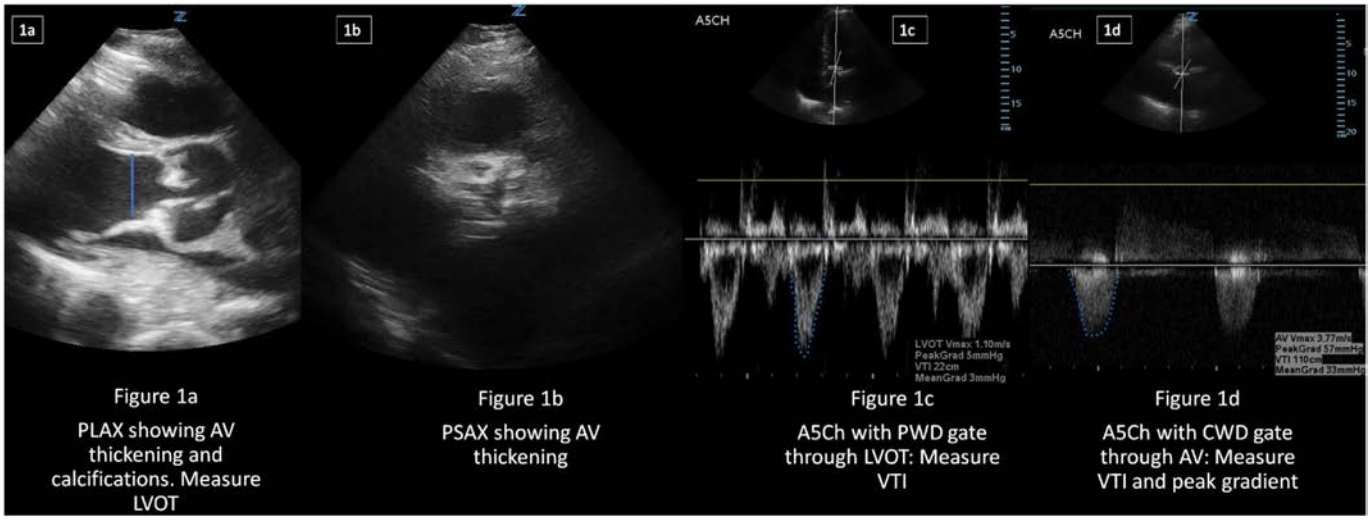


Fig. 1. POCUS evaluation of severe aortic stenosis.

maximum blood flow velocity through the AV and is normally <2.5 m/s. The mean transaortic gradient is the pressure difference between the LV and aorta in systole, normally <20 mmHg. All three physiologic parameters can be obtained using the cardiac preset with the machine to solve the continuity equation. Stroke volume (SV) is obtained by calculating the cross-sectional area (CSA) of a structure and the flow over time through that structure (VTI). The heart's stroke volume is derived from the CSA and the associated VTI. Since the volume of blood passing through the AV and LVOT volume should be consistent, the continuity equation is used to calculate AV_{area} if an AV VTI is also obtained: CSA_{LVOT} x VTI_{LVOT} = AV_{area} x VTI_{AV}, sometimes also referred to as A1 x V1 = A2 x V2, respectively. To obtain these values, use the cardiac preset to obtain an apical five-chamber (A5CH) view focusing on the AV. First, obtain AV measurements with continuous wave Doppler (CWD) through the AV. Trace the downward systolic waveform to calculate the AV VTI and peak aortic velocity (V_{max}). Switch to pulse wave Doppler (PWD) in the same A5CH view and place the Doppler gate through the LVOT. Trace the obtained waveform in the same fashion to calculate the LVOT VTI. Finally, switch to PLAX and the LVOT diameter is measured in midsystole to calculate the LVOT CSA (Fig. 1b). If the machine does not automatically compute the CSA, it can be calculated using the following equations:

$$\text{LVOT CSA} = (\pi \times \text{LVOT diameter}\hat{}2)/4.$$

Or

$$\text{LVOT CSA Simplified equation} = \text{Diameter}\hat{}2 \times 0.785.$$

Utilizing the report feature (machine-specific), the AV_{area}, V_{max}, and mean transaortic gradient will be displayed; if such a package is not available, the algebraic equation to solve for the AV_{area} is as follows:

$$A_2 = (A_1 \text{ x } V_1)/V_2.$$

or

$$AV = (CSA_{LVOT} \text{ x } VTI_{LVOT}/VTI_{AV}).$$

Figures 1c, d, and 2 provide concise details and diagrams for replication. An AV_{area} < 1.0 cm^2, peak aortic velocity >4 m/s, and mean transaortic gradient >40 mmHg is diagnostic for severe aortic stenosis.

3.1.4. Additional guidance

In cases of depressed ejection fraction, an AV_{area} < 1.0 cm^2 can be the only abnormal parameter. This is termed a "low flow, low gradient" state. In these cases, the ventricle is too weak to create enough stroke volume to create a significant pressure gradient or velocity despite the AV being stenotic. A diagnostic tool in these cases is the dimensional index. Dimensionless Index (DI) = LVOT VTI /AV VTI; a simplified and reasonably accurate approach is just using the Vmax of each (DI = LVOT Vmax/AV Vmax). A value <0.25 is indicative of severe AS [19]. To ensure the gradient measurements reflect AS (rather than sub- or subra-valvular stenosis), color flow can be added to confirm the origin of the stenosis (area of highest aliasing) is at the level of the aortic valve. While not commonly used in comprehensive echocardiograms, it is a simple addition that may help the point-of-care user determine whether the obstruction is in the expected location.

3.1.5. Pitfalls

[20] Small measurement changes can greatly impact the accuracy of the classification and results (refer to the algebraic equation above). A common issue is the LVOT diameter measurement where small inaccuracies can cause significant value changes and misinterpretation; ensuring midsystolic portion of cardiac cycle, measuring inner-to-inner, zooming in on area of interest and multiple measurements can all improve accuracy [20]. There is some debate where the most specific site to measure relative to the aortic valve annulus, but majority of guidelines are around 5 mm from the annulus or at the annulus itself. [21-23]

Other potential pitfalls from measurement inaccuracies also exist. When obtaining the LVOT VTI (V1), not having the Doppler gate appropriately positioned can underestimate AV_{area}. Lastly, when measuring aortic valve VTI, if the Doppler misses the narrowed aortic valve orifice, a lower-than-expected number will result; this will give an overestimated, less severe than actual AV_{area}. Color Doppler can help identify the best location to place the Doppler gate.

High gradients across the aortic valve do not only signify aortic stenosis; one should also consider conditions such as LVOT obstruction, supravalvular stenosis, and subvalvular stenosis; a normal appearing aortic valve with high velocities may help point towards one of these other conditions, and also using color Doppler as described above [20].

3.2. Infective endocarditis

3.2.1. Description

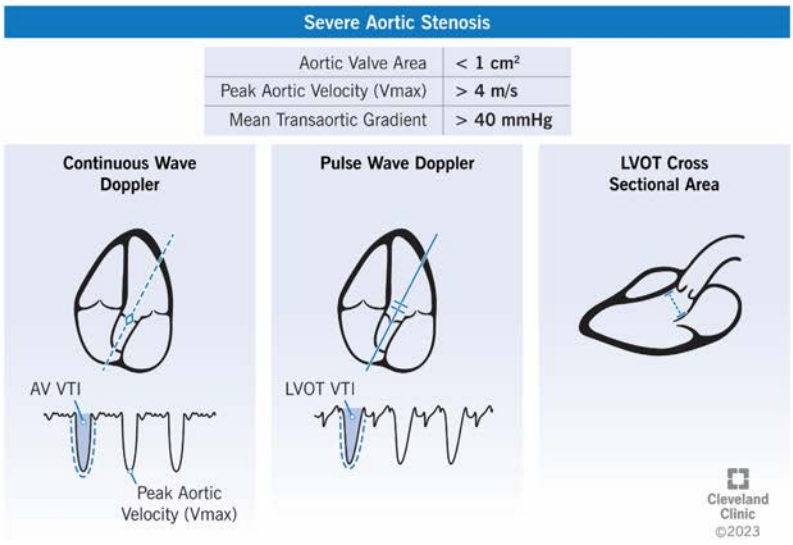
Infective endocarditis (IE) is a medical emergency with an in-hospital mortality between 15 and 30 % [24]. IE presents a diagnostic challenge as the presenting signs and symptoms are nonspecific, and classic physical exam findings are rare [24]. Criteria commonly used for diagnosis, such as those in the Duke Criteria, are unavailable in the emergency department and not useful for EPs. Risk factors are driven by turbulent flow or structural abnormalities, with mitral valve prolapse being an intermediate risk factor for IE [24]. More than 70 % of cases occur in native valves, with almost 80 % of cases involving the mitral or aortic valves; however, right-sided endocarditis is more likely to occur in the setting of intravenous drug abuse or intravascular devices [25]. Numerous organisms are responsible, the most common being *Staphylococcus aureus*, *Streptococcus viridans*, enterococci, and coagulase-negative staphylococci [25]. Point-of-care ultrasound (POCUS), despite its subtleties and limits, offers a practical approach for EPs to evaluate suspicion of endocarditis. The focused questions include assessing obvious vegetations and secondary findings, such as regurgitation and heart failure.

3.2.2. Evidence

Given that echocardiography findings comprise the major criteria for diagnosis, transthoracic echocardiography (TTE) is used for initial evaluation [25]. Documented sensitivity and specificity of TTE for diagnosis on native valves is ~70 % and 80–90 %, respectively [25,26]. However, the sensitivity of TTE is lower with prosthetic valves (50 %). Because of the sensitivity of TTE, transesophageal echocardiography (TEE) is recommended with a negative TTE unless the patient is low risk; the sensitivity for native and prosthetic valves are 96 % and 92 %, respectively [24]. The tricuspid valve is most commonly affected with right-sided endocarditis [2]. However, in some studies, TTE is as sensitive as TEE for diagnosing tricuspid valve endocarditis, given the anatomy of the right-sided valves [27].

While IE is commonly diagnosed with a comprehensive echocardiogram, there have been numerous reports of POCUS-guided diagnosis of right-sided infective endocarditis by TTE, even by handheld devices [28-33]. Despite the reduced sensitivity of TTE, left-sided endocarditis has also been previously noted to have been diagnosed by POCUS [33-40]. A case of pediatric IE has additionally been documented with POCUS[41]. In some of these reports, POCUS was used to identify complications, including valvular regurgitation, pulmonary infarction with empyema, heart failure, and regional wall motion abnormalities [32,33,38-41]. Although there have not been many studies evaluating POCUS in the diagnosis of IE, one study did show sensitivity

A. Severe Aortic Stenosis Illustrated Infographic



B. Severe Aortic Stenosis Diagnosis Reference Card

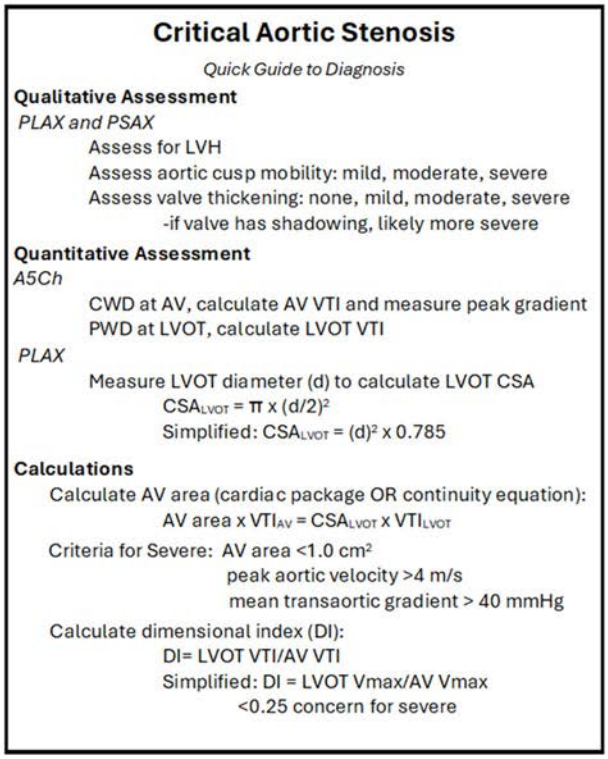

Critical Aortic Stenosis
Quick Guide to Diagnosis
Qualitative Assessment
PLAX and PSAX
Assess for LVH
Assess aortic cusp mobility: mild, moderate, severe
Assess valve thickening: none, mild, moderate, severe
-if valve has shadowing, likely more severe
Quantitative Assessment
A5Ch
CWD at AV, calculate AV VTI and measure peak gradient
PWD at LVOT, calculate LVOT VTI
PLAX
Measure LVOT diameter (d) to calculate LVOT CSA
CSALVOT = π x (d/2)²
Simplified: CSALVOT = (d)² x 0.785
Calculations
Calculate AV area (cardiac package OR continuity equation):
AV area x VTIAV = CSALVOT x VTILVOT
Criteria for Severe: AV area <1.0 cm²
peak aortic velocity >4 m/s
mean transaortic gradient > 40 mmHg
Calculate dimensional index (DI):
DI= LVOT VTI/AV VTI
Simplified: DI = LVOT Vmax/AV Vmax
<0.25 concern for severe

Fig. 2. Severe aortic stenosis.

of 77 % and specificity of 94 % of POCUS compared to traditional echocardiography, which included some patients with prosthetic valves [42].

3.2.3. Assessment: procedure and evaluation

Beyond the cardinal views comprised of the parasternal long axis (PLAX), parasternal short axis (PSAX), apical 4/5 chamber (A4C, A5C), and subcostal (SC), the right ventricular inflow view (RVI)can also be helpful to evaluate the tricuspid valve (Fig. 3a). The RVI, also called right ventricular inflow tract (RVIT) view can be obtained from the PLAX by fanning the probe towards the right hip. Color flow can be employed, particularly in the PLAX and A5C, to assess for secondary regurgitant jets.

The PLAX provides a view of the aortic and mitral valves and can be useful to evaluate for regurgitant jets. The mitral level of the PSAX can be used to assess both the mitral and tricuspid valves. The aortic level of the PSAX is another window to evaluate the aortic valve; one can also view the pulmonary valve at this level. The SC view can be used to assess the mitral and tricuspid valves.

There are three TTE findings comprising major criteria: vegetation, abscess or pseudoaneurysm, and new dehiscence of a prosthetic valve [24]. Vegetations are echogenic masses on endocardial structures or

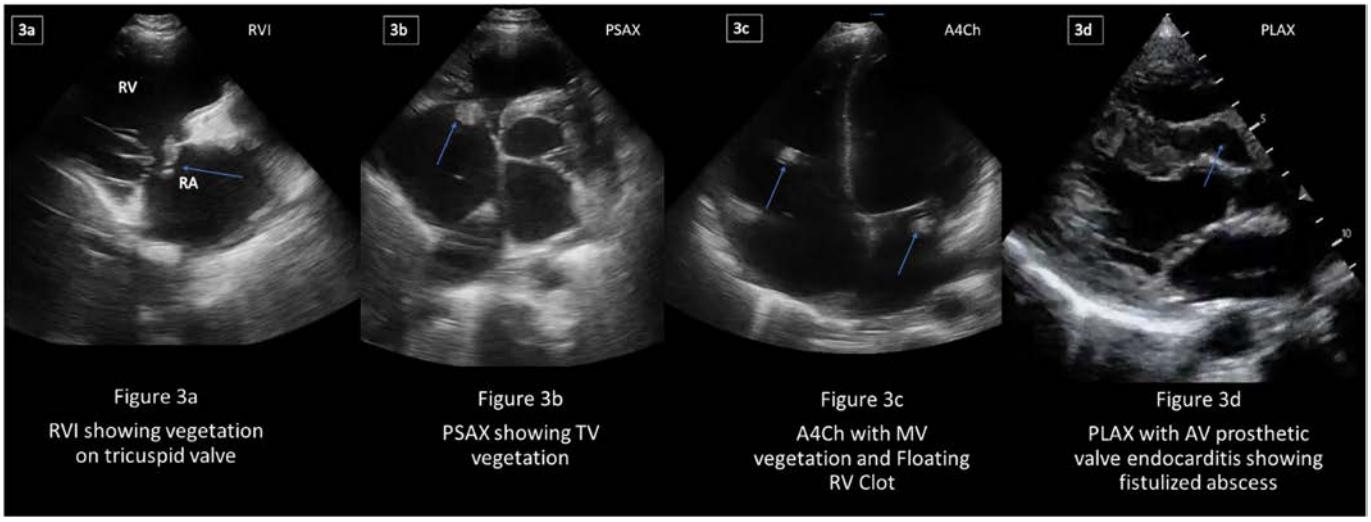


Fig. 3. POCUS evaluation of infective endocarditis.

implanted material that have erratic movement independent of the valve and are typically on the upstream (atrial) side (Fig. 3b and c) [24,27]. Vegetations from infective endocarditis are often difficult to differentiate from other causes: marantic vegetations (sterile vegetations from multiple causes), myxomatous valve disease, cusp prolapse, valve fibroelastoma, and Lambl's excrescences (rare fibrotic structures that can embolize). Abscess and pseudoaneurysm can be identified as perivalvular hypoechoic cavities (Fig. 3d). These are differentiated by color Doppler, where pseudoaneurysm will have color-Doppler flow and abscess will not [24]. *S. aureus* and *S. lugdunensis* tend to be more aggressive and are more likely to form perivalvular abscess [25]. An echo finding of prosthetic valve dehiscence is paravalvular regurgitation that may have a rocking motion of the prosthesis [24].

Additional echo findings of IE include perforation, fistula, valve aneurysm, and valve regurgitation [24,25]. Perforation displays interruption of endocardial tissue, and the fistula will have color-Doppler flow between the two neighboring cavities with the perforation (Fig. 4). Saccular bulging of valvular tissue indicates valve aneurysm. Valve regurgitation may result from mitral chordal rupture, leaflet rupture, leaflet perforation, or interruption of valve coaptation. Early prosthetic valve endocarditis usually affects the junction of the annulus and sewing ring with perivalvular abscess, dehiscence, pseudoaneurysm, and fistulae (Fig. 4). Late prosthetic valve endocarditis more commonly affects the leaflets, typical of native valve endocarditis [24].

3.2.4. Additional guidance

Some of these findings can also aid in predicting a complicated course and deciding the need for surgery. These include large vegetations (>10 mm) as they are more likely to embolize and are associated with greater risk for abscess or pseudoaneurysm, severe valve regurgitation, valve perforation, and decompensated heart failure [2]. Heart failure is the most common complication and indication for surgery; heart failure is more commonly present when IE affects the aortic valve [1].

3.2.5. Pitfalls

TTE sensitivity is limited when evaluating for vegetations or with complications of endocarditis such as pseudoaneurysm or abscess; TEE has higher sensitivities for these; however, due to the anatomy of the heart relative to the chest wall, TTE still performs well when evaluating for right-sided vegetations. Calcifications often bring another challenge in diagnosing vegetations. Additionally, other conditions may appear similar to IE on imaging including merantic vegetations, chiari network, redundant eustachian valve, myxomatous changes, lambes excrescence, etcetera [43]. This may misguide the treatment course. Applying the clinical scenario, reviewing prior echo studies, assessing for complications of endocarditis, and ordering a comprehensive echocardiogram help the provider differentiate between these conditions.

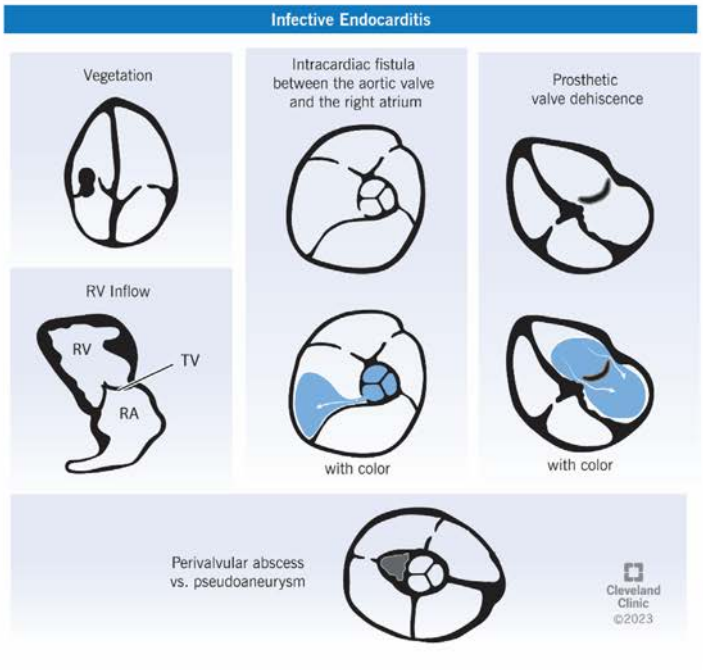
3.3. Left ventricular outflow tract obstruction

3.3.1. Description

Left Ventricular Outflow Tract Obstruction (LVOTO) is one of the common causes of unexplained hypotension in critically ill patients [44-47]. LVOTO incidence is underestimated and missed due to a lack of clinical suspicion, particularly in patients without a documented history of hypertrophic cardiomyopathy (HCM). The presence of severe shock in the setting of normal or hyperdynamic ejection fraction should raise suspicion about the potential for LVOT obstruction. It is important for LVOTO to remain on the differential diagnosis, specifically in patients with refractory hypotension, as appropriate therapy may be starkly different than usual measures. Patients with LVOTO can have severe hypotension, pulmonary edema, and a clinical picture consistent with cardiogenic shock. Within this clinical picture, it is common to administer diuretics and place the patient on vasopressors, which can ultimately worsen LVOTO and cause worsening LV function and perfusion [48,49]. Recent literature suggests that along with volume resuscitation to supplement preload, pure vasopressors such as phenylephrine or vasopressin, as well as beta-blockade, may be indicated, with the goals of increasing afterload, improving LV filling, and minimizing obstruction [44-47,49]. Appropriate treatment of LVOTO requires a prompt diagnosis, which can be accomplished through POCUS.

Although commonly described in the setting of HCM, LVOTO is a more general term that refers to any mechanism that limits blood flow out of the LVOT [33,37]. HCM has traditionally been used to depict LVOTO, given the septal hypertrophy and systolic anterior motion (SAM) of the anterior mitral valve leaflet, contributing to LVOTO [39]. However, recent literature has identified more dynamic causes of LVOTO, even in the setting of mild structural defects [34-36,38]. Several structural pathologies can cause LVOTO, including aortic stenosis, subaortic and supra-valvular aortic stenosis, coarctation of the aorta, and others [37]. These structural pathologies can combine with critical illness states, such as a hyperdynamic/underfilled LV, and cause dynamic LVOTO [34,38]. Patients with distributive or septic shock, hypovolemic shock, or those undergoing inotropic therapy may be especially susceptible [34-36,38].

A. Infective Endocarditis Illustrated Infographic



B. Infective Endocarditis Diagnosis Reference Card

Infective Endocarditis

Quick Guide to Diagnosis

Qualitative Assessment

Vegetations: echogenic masses with independent motion on the atrial side of the valve.

Look for complications:
- Regurgitation with color doppler
- Perivalvular abscess and pseudoaneurysm
  - Hypoechoic perivalvular cavity
    - Flow: pseudoaneurysm
    - No flow: abscess
- Saccular aneurysm will show saccular bulging
- Perforation: disruption of endocardial tissue between cavities
- Fistula shows flow between neighboring cavities

PLAX: Assess mitral/aortic valves
Fan upwards toward right hip to get RV Inflow view
Assess tricuspid

PSAX: Assess mitral/tricuspid valves (mitral level)
Assess aortic/pulmonic valves (aortic level)

A4Ch: Assess mitral/tricuspid valves

A5Ch: Assess aortic valve

SC: Assess mitral/tricuspid valve

Fig. 4. Infective endocarditis.

3.3.2. Evidence

Although sonographic evaluation has long been used to guide the diagnosis and treatment of acute LVOTO, there is minimal literature with which to create an evidence-based standard [34-36]. This lack of data is partly due to the dynamic, often multifactorial etiology of LVOTO, which requires several types of management. For example, while there is a large pool of research about the management of HCM, evidence-based standards for POCUS-driven recognition and treatment of acute

LVOTO, whether secondary to HCM or other etiologies, are lacking. The following details the most up-to-date and accurate methods to diagnose acute LVOTO.

3.3.3. Assessment: procedure and evaluation

Sonographically, LVOTO is diagnosed with a measured peak gradient of at least 30 mmHg through the left ventricular outflow tract via continuous wave Doppler (CWD) in the apical 5-chamber (A5Ch) using the modified Bernoulli eq. ($4 \times V^2_{max}$), which most modern ultrasound machines calculate automatically (Fig. 5a) [45,48-50]. Severe LVOTO is denoted by a peak gradient of 50 mmHg or more, which requires emergent intervention in the setting of shock [46,49,50].

A comprehensive, step-wise approach to obtain this measurement begins with the parasternal long axis (PLAX) view [4,8] usually in [8]. In this view, one can assess for hyperdynamic left ventricular (LV) function, and a mid-systolic measurement of the LVOT diameter (mean ~2 cm) may also be obtained to evaluate for narrowing (Fig. 5b) [51,52]. Additionally, evidence of SAM may be visualized on PLAX view, as the high velocities across the LVOT decrease pressure on the anterior mitral valve leaflet, pulling it into the LVOT during systole, known as the Bernoulli Effect(Fig. 5c) [44,52,53]. If concerned specifically for HCM as the underlying cause of SAM/LVOTO, unexplained ventricular wall thickness > 15 mm with a septum-to-posterior wall thickness ratio of 1.3 or higher is diagnostic; these are best performed during diastole, using PLAX or parasternal short axis (PSAX) views [48,50,52,53].

Next, an apical 5-chamber (A5CH) view should be obtained, with good visualization of the LVOT. Firstly, color flow Doppler can be utilized to assess for high velocities in the LV cavity and LVOT which are signaled by aliasing. Aliasing on color Doppler will appear as bright colors at the extremes of the color map. The ultrasound is then placed in pulse wave Doppler (PWD) mode, where sequential measurements are obtained throughout the left heart chamber. This is performed by placing the PWD gate over several areas, including the mid-cavitary region(s), the aortic valve, and the LVOT, to compare flow velocities at these specific points (to assess for the point of aliasing signaling high velocities) With true LVOTO, aliasing will be seen on PWD most significantly at the LVOT region. To recognize aliasing on spectral Doppler, note the waveform will appear to "wrap around" from one side of the baseline to the other side (appearing as columns) indicating velocities are higher than the Nyquist limit, and concern for obstruction of flow (Figs. 5d, 6). Once identified with the Doppler gate held on the LVOT, the mode should then be switched to continuous wave Doppler (CWD) to quantify the highest velocity flow, revealing "dagger-shaped" waveforms, which are used to calculate the measured peak gradient (Fig. 5a). This completes the necessary components of the bedside ultrasound evaluation, with gradients >30 mmHg confirming the diagnosis of LVOTO [45,49,50].

3.3.4. Additional guidance

Acute LVOTO differs from the valvular emergencies listed in this review in that it is always a secondary process—it can manifest as a complication of multiple underlying pathologies. Ultrasound is an excellent tool that can aid in stabilizing a patient in the emergent setting and monitoring the extent of obstruction as the patient undergoes appropriate treatment. Of course, it is necessary to acknowledge that the underlying cause of LVOTO must be addressed to provide definitive care, which may not be possible using POCUS alone.

3.3.5. Pitfalls

The diagnosis of LVOTO can be complicated by patient pathophysiology. Measurement of the mitral regurgitation signal rather than the outflow tract gradient may occur as they both occur during systole and blood is flowing out of the ventricle. Ensuring the appropriate Doppler location and correcting the Doppler angle along the flow of the LVOT can help the operator overcome these limitations. Additionally, it can be difficult to determine the etiology of the gradient [54]. Lesions that cause a fixed obstruction of the LVOT (such as aortic stenosis) will have a more parabolic waveform on CWD and will not have the dagger-like waveform as seen with dynamic LVOT obstruction.

3.4. Mitral stenosis

3.4.1. Description

Mitral Stenosis (MS), a disease largely diagnosed by echocardiogram and intervenable by medication and/or valvuloplasty, can present with many common complications seen in the emergency department (ED), including heart failure, thromboembolic disease, atrial fibrillation, and pulmonary hypertension [55,56].

Alone or in combination with other valvular abnormalities, MS is usually related to a history of rheumatic heart disease [57]. However, only 50–70 % of patients with MS report a history of rheumatic heart disease [58-60]. Additional causes of MS include mitral annular calcification, radiation-associated valvular disease, systemic rheumatic diseases such as lupus or rheumatoid arthritis, and rarely congenital abnormalities [60,61]. MS results when there is decreased commissural

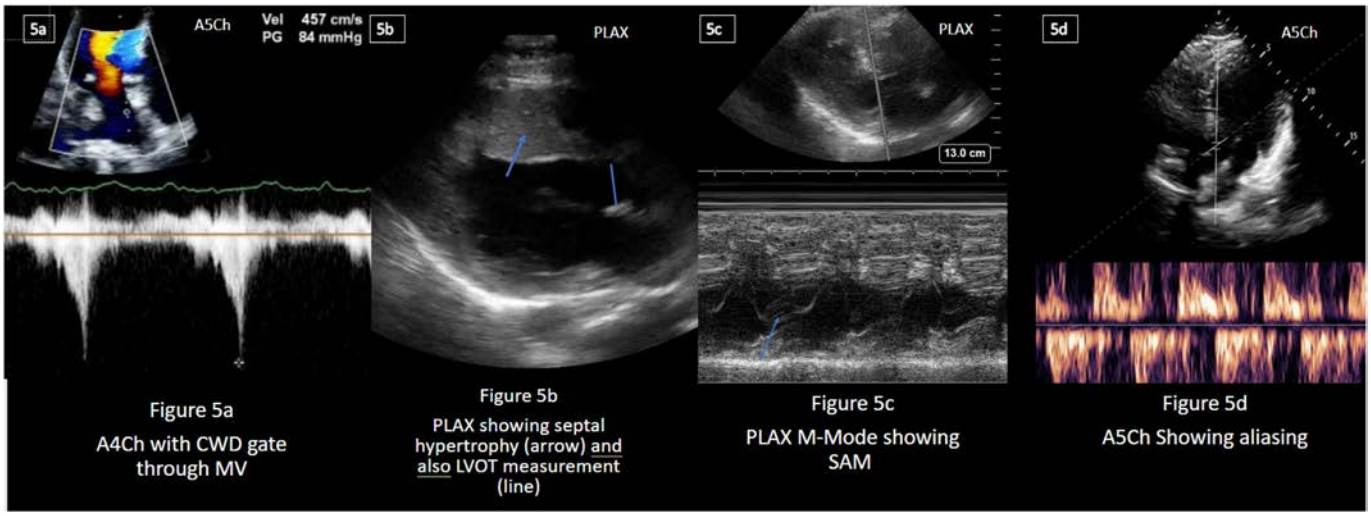
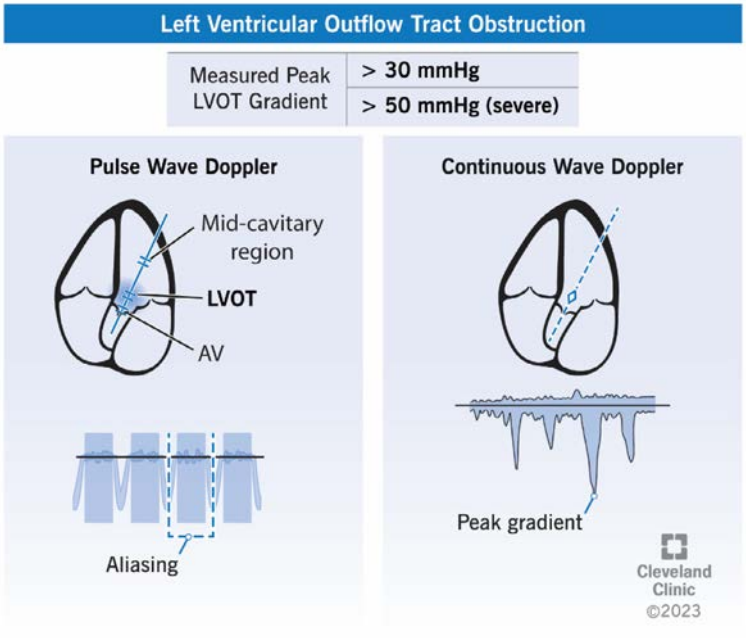


Fig. 5. POCUS evaluation of left ventricular outflow tract obstruction.

A. Left Ventricular Outflow Tract Obstruction Illustrated Infographic



B. Left Ventricular Outflow Tract Obstruction Diagnosis Reference Card

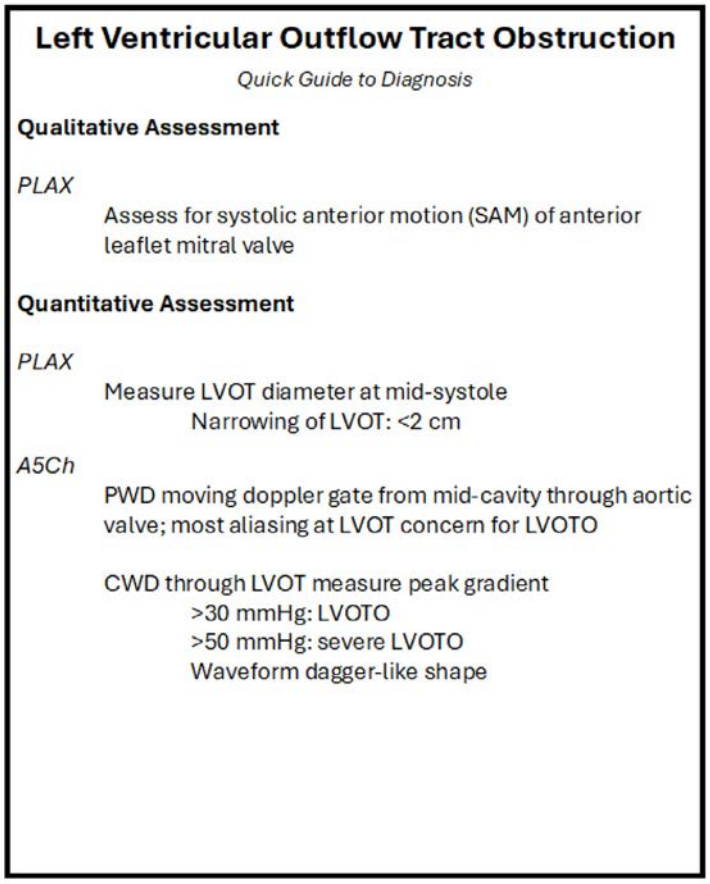


Fig. 6. Left ventricular outflow tract obstruction.

separation due to partial fusion and decreased mobility of the valve leaflets; if left untreated, it can progress to a hemodynamically significant lesion. Point-of-care ultrasound (POCUS) evaluation of the mitral valve (MV) can be achieved using the traditional 2-D, M-Mode, and Doppler echo. Additional adjuncts less likely to be available/reasonable in the ED include TEE and 3-D echo.

3.4.2. Evidence

There is little literature on the use of POCUS in mitral stenosis. Even cardiology-based literature has little data on the sensitivity, specificity, or diagnostic accuracy of comprehensive echocardiography given the absence of alternate diagnostic modalities; most of these studies are compared to physiologic parameters or surgical specimens. However, case reports highlight POCUS leading to comprehensive ultrasound and appropriate management. A recent prospective study evaluated cardiologists performing POCUS in the clinic for valvular conditions before a comprehensive echo. It showed good sensitivities and specificities for different valvular conditions (82.4–100 % sensitive and 96.7–100 % specific); there was one diagnosis of mitral stenosis out of the 77 patients [62,63].

3.4.3. Assessment: procedure and evaluation

In PLAX, the stenosis causes the valve to bulge into the left ventricle during diastole (doming) and creates a knee-bend or hockey-stick appearance of the anterior leaflet (Fig. 7a) While in PLAX, assess for leaflet immobility, leaflet calcifications and thickening, and subvalvular thickening; there are grading scores used that were left out due to complexity (Wilkins and Cormier scores). Typically, increasing calcifications/thickness and restriction of leaflets correlate with more advanced disease [64,65].

Left atrial enlargement (LAE) is almost always seen in the setting of mitral stenosis. LAE can be qualitatively compared to the aorta and the right ventricular outflow tract for enlargement in PLAX. Quantitatively, the diameter can be assessed in PLAX during end-systole, with abnormal being greater than 4 cm; however, the more accurate measurement is the left atrial volume index (LAVI), with abnormal being >34 ml/m^2 [66]. This is obtained by tracing the chamber size, best by combining the apical four-chamber with the apical two-chamber with a Simpson approach and indexing to body surface area [67].

In PSAX, one can appreciate the restricted motion and fusion of the commissures (corners where the two leaflets meet). While in PSAX, calculate the mitral valve area with planimetry, which is directly tracing the area of the valve opening (Fig. 7b). This is often the primary way to assess severity, tends to be quite intuitive for point-of-care providers, and has shown the greatest correlation when echo was compared to explanted valves in some studies [68-70]. First, find the optimal view of the valve orifice, which should be perpendicular to the probe and have an elliptical appearance. Next, the valve is assessed during maximum excursion (usually mid-diastole). Most machines then allow one to trace the orifice to provide a cross-sectional area. In the setting of atrial fibrillation, multiple calculations are likely to be required for accuracy [67]. One other consideration, in the setting of severe calcification, is to adjust gain down to avoid exaggerated measurements.

The cross-sectional area of a normal MV is 4–6 cm2. When the valve area is <2.5 cm2, symptoms such as dyspnea on exertion result. A valve area of 1.0–1.5 cm2 is considered moderate stenosis and may result in rest symptoms [68]. Severe MS, area < 1 $cm^{2,}$ may lead to decreased preload and thus decreased stroke volume and cardiac output.

Doppler is the last primary piece used to assess MS. The two primary measurements, taken from the same view and same spectral Doppler, are the mean gradient and the diastolic half-time. After obtaining an apical four-chamber, apply Continuous Wave Doppler (CWD) through the mitral valve inflow area, producing the familiar E wave and A wave (as in diastology assessment). Then, by tracing the mitral flow envelope (the whole E and A waves together) or MV VTI, the machine should calculate the mean gradient (Fig. 7c). Note that three consecutive waveforms should be traced and averaged in the setting of atrial fibrillation [56]. In severe disease, the mean gradient should be at least >5 mmHg, if not 10 mmHg, but because it is very dependent on heart rate and flow, it was not included in the 2020 ACC/AHA Guidelines for staging MS; further, the guidelines were updated where traditionally 5–10 mmHg was moderate and > 10 mmHg was severe [67,70]. In the same view, the downslope of the E wave from the peak is traced to get the pressure half-time, or diastolic pressure half-time (Fig. 7d, Fig. 8). This is the time it took for the peak pressure to reduce by half; the machine typically will calculate this. With severe disease, it is typically >150 ms, and very severe >220 ms (with moderate being 100-149 ms); however the measurement is also dependent on many factors [68-70]. By taking 220/pressure half-time, you can also estimate valve area, which correlates well with invasive assessments [71].

3.4.4. Additional guidance

If one already has obtained or subsequently obtains the LVOT VTI and using the MV VTI, the dimensionless mitral stenosis index (DMSI) can be calculated with this equation:

DMSI = (LVOT VTI/MV VTI). This simple calculation has shown that a result of 0.35 to 0.50 is consistent with severe MS, and < 0.35 suggests very severe MS[72].

There are multiple other helpful Doppler measurements, but they add considerable time and complexity and are not discussed in detail. Color Doppler can assess for secondary mitral regurgitation, which largely has interventional implications if present [71,72]. M-mode may aid in diagnosis and severity of MS, but is intricate, and is not one of

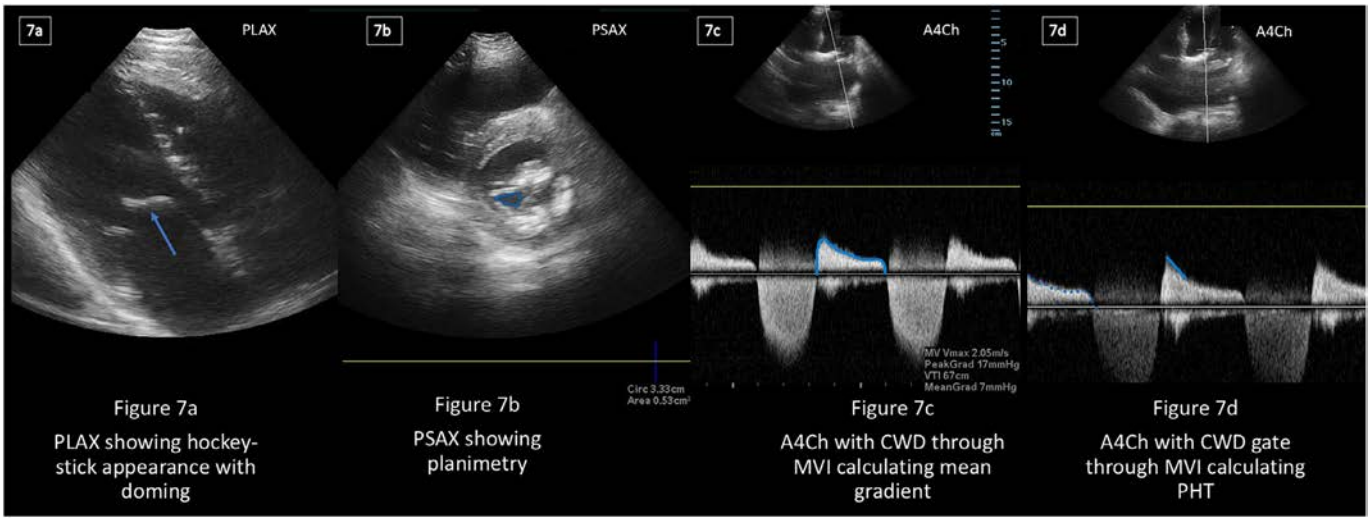
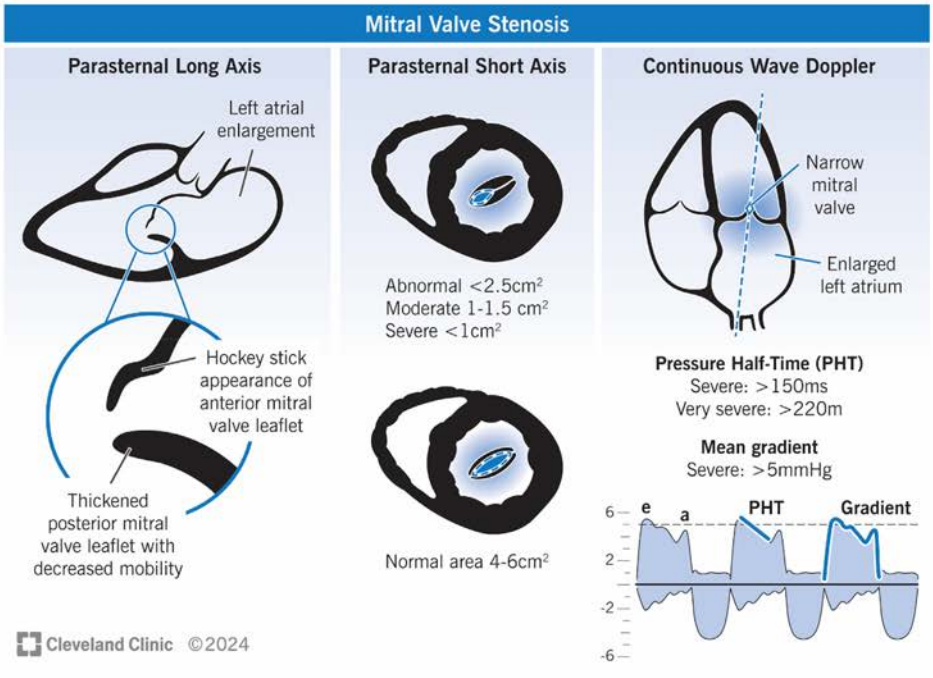


Fig. 7. POCUS evaluation of mitral stenosis.

A. Mitral Stenosis Illustrated Infographic



B. Mitral Stenosis Diagnosis Reference Card

Mitral Stenosis

Quick Guide to Diagnosis

Qualitative Assessment

PLAX

Observe for diastolic doming or hockey stick appearance of anterior leaflet
Assess for leaflet immobility, leaflet calcifications/thickness, and subvalvular thickening

Quantitative Assessment

PLAX

Assess for LAE: >4cm diameter abnormal.
More accurate tracing border, especially doing biplane

PSAX, mitral level

Measure mitral valve area with planimetry maximum excursion (usually mid-diastole), multiple measure ments if atrial fibrillation, drop gain with calcifications

Normal area 4-6cm2,
Abnormal <2.5 cm2,
Moderate 1.5-1 cm2,
Severe <1 cm2

A4Ch

CWD through MV inflow, trace envelope
Obtain Mean gradient by tracing diastolic inflow envelope; very heart rate dependent
Severe: >5mmHg
Obtain Pressure Half-time/Diastolic Pressure Half-time by tracing downslope after the peak of the E wave
Severe >150ms, Very severe >220ms

Fig. 8. Mitral stenosis.

the cardinal means of such in the latest guidelines. Lastly, discrepancies of prior Doppler measurements can be assessed by other means to calculate the mitral valve area (continuity equation and proximal velocity surface area or PISA) [68,69].

Due to the pathophysiology, patients with severe MS often have secondary pulmonary hypertension; if familiar with obtaining a right ventricular systolic pressure (RVSP) or a pulmonary arterial systolic pressure (PASP), it is typically above 50 mmHg in severe disease [72]. It is also worth noting severe stenosis can cause right heart and IVC dilation while the LV pressures remain low. Clinically, this may lead to challenging preload assessments as diuresis can cause hypotension with severe MS [68,69].

3.4.5. Pitfalls

While there are multiple methods for imaging the MV, it remains subject to both overestimation (Severe Aortic Regurgitation, ASD) and underestimation (abnormal left ventricular relaxation, excessive gain) of the valve area. Thus, determination of the presence and degree of MS can be difficult. Multiple modalities can be used in concert, but the assessment of MS should include, at minimum, measurement of mitral valve area by planimetry, pressure half time, and mean pressure gradient.

Additionally, ensuring the measurement of the mitral valve area is at the leaflet tips for planimetry measurement can be challenging with standard 2D imaging. Due to the doming of the leaflets, it is easy to overestimate the MVA with planimetry; cardiology often uses x-plane, bi-planeor 3D modalities to ensure the measurement occurs at the leaflet tips.

The pressure half-time is influenced by many factors, such as decreased left atrial compliance, LVH, or other valvular abnormalities such as AR or severe MR. It is also important for EPs to remember that pressure gradient, while valuable in assessment, is highly dependent on heart rate and cardiac output. Despite the potential for both overestimation and underestimation of the mitral valve area, MS remains an entity that can be identified using bedside echocardiography and potentially influence decision-making in the ED.

3.5. Mitral regurgitation

3.5.1. Description

Acute mitral regurgitation (MR) is a medical and time-sensitive surgical emergency in which patients can present gravely ill with acute respiratory failure from pulmonary edema and/or cardiogenic shock. Acute MR may be misdiagnosed as a pulmonary condition or diagnosed as decompensated congestive heart failure. Additionally, physical exam findings and history related to a valvular emergency may be subtle or not present. For example, approximately 50 % of patients with moderate to severe acute ischemic MR have no audible cardiac murmur [73]. Early echocardiography has a high potential to confirm or exclude acute severe MR and thus alter patients' course and outcomes. For these reasons, early comprehensive trans-thoracic echocardiography (TTE), if available, should strongly be considered in presentations of acute severe pulmonary edema and/or cardiogenic shock.

The mechanism of MR can be classified as primary, an abnormality intrinsic to the mitral valve (MV) leaflets, or secondary, a cause extrinsic to the MV, such as left ventricle/left atrium (LV/LA) remodeling. Focusing on the presentation of acute native valve MR and whether the patient will require surgical treatment, it may be more practical for the acute care provider to classify the etiology into (1) Ischemic MR, (2) Non-ischemic MR, and (3) acute decompensation of chronic MR. Different mechanisms are responsible for MR in prosthetic valves and are not covered here.

Ischemic MR can present with a ruptured papillary muscle, which may be fatal [74]. Impending papillary muscle may present as new leaflet prolapse in the setting of cardiac ischemia [74]. Papillary muscle displacement can also occur due to regional wall motion abnormalities, which can cause asymmetric leaflet tethering and acute MR [73,74].

Nonischemic MR can be seen secondarily from LV/LA remodeling in nonischemic cardiomyopathy and/or widening of the MV annulus, causing reduced coaptation and regurgitant orifice. It can also be seen primarily in myxomatous disease/mitral valve prolapse, degenerative changes, rheumatic heart disease, congenital causes, mitral valve perforation, infective endocarditis, and even spontaneous rupture of the mitral valve. MR can also be seen in settings of dynamic LVOT obstruction and stress cardiomyopathy [73].

Acute decompensation of chronic MR can occur due to the progression of underlying disease, an acute cardiogenic cause, or a noncardiogenic stressor. It may present in the setting of known MR or may be the first presentation of chronic MR if unknown or untreated.

3.5.2. Evidence

Numerous studies and guidelines outline the recommendations for the echocardiographic assessment of chronic mitral regurgitation [75-78]. Many of the principles in these guidelines apply to the echocardiographic assessment of acute MR. There have been many documented cases of the use of POCUS in the diagnosis of acute MR [79-83]. Still, there are no uniform recommendations for point-of-care providers to assess acute MR. If available and possible, comprehensive TTE is recommended. If not readily available or possible, point-of-care ultrasound can be performed to assess for 2D (B-mode) findings, color Doppler, pulsed-wave Doppler (PWD), and continuous wave Doppler (CWD) plus or minus quantitative Doppler assessment of mitral regurgitation.

3.5.3. Assessment: procedure and evaluation

The critical findings one should assess in a two-dimensional view are signs of papillary muscle rupture and flail leaflet. Papillary muscle rupture will be seen on echo as the classic round or triangular mass (the papillary muscle head) swinging back and forth between the left atrium and ventricle with the entire leaflet prolapsing into the left atrium during systole (Fig. 9a). Impending papillary muscle rupture may present as new leaflet prolapse in the setting of cardiac ischemia [74,84]. Papillary muscle displacement can occur due to regional wall motion abnormalities, which can cause asymmetric leaflet tethering, resulting in acute MR [85,86]. Symmetric leaflet tethering tends to occur from non-ischemic cardiomyopathy. This will appear as a characteristic 'tenting' of the leaflet and reduced coaptation. (See Fig. 10.)

A flail leaflet will extend behind (towards the atria) the non-ruptured leaflet where it often lies parallel (parallel sign). Normal coaptation will not occur, and one may see the flail leaflet moving into the LA during systole. One also may see chordal structures in the atrium. Attention must be taken to optimize visualization, including a zoomed-in view of the valve and valve apparatus.

In addition to qualitatively assessing the MV, one should assess left heart size and function to delineate the etiology and chronicity. Acute MR will typically have a normal LV and LA size (due to lack of time to compensate), and the LV will often be hyperdynamic in efforts to increase stroke volume. In chronic MR, there is often progressive LV and LA dilation as well as progressively worsening LV systolic dysfunction.

Color flow Doppler is a key means of MR evaluation and is primarily assessed in Parasternal Long Axis (PLAX) and Apical 4-chamber (A4CH) views by placing the color box over the LA and the inflow portion of the LV. A regurgitant jet is visualized as blood flows away from the transducer during systole. The area and angle of the jet can provide further details. While jet area is imprecise in several studies and likely underestimates severity in eccentric jets, it is easy to perform [75,87,88]. If >50 % LA is involved or the eccentric jet wraps around the LA, it is likely severe. In the setting of a flail leaflet, the eccentric jet typically points away from the leaflet at an acute angle (often <45 degrees). Therefore, an anterior flail leaflet will often have a posterolateral eccentric jet, and a posterior flail leaflet tends to have an anteromedial jet (Fig. 9c).

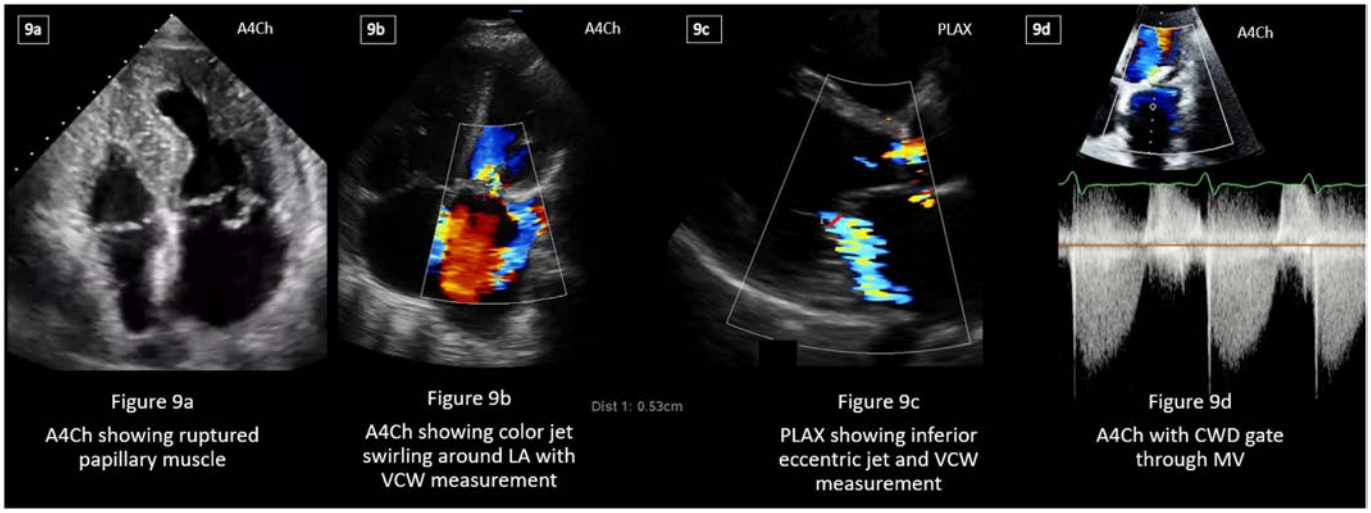


Fig. 9. POCUS evaluation of mitral regurgitation.

While assessing the regurgitant jet with color, one can also appreciate the proximal flow convergence (PFC) and the *vena contracta* width (VCW). The PFC is noted as the color flow on the ventricular side of the valve converging proximal to the regurgitation. Reliable measurement is challenging and is not covered here, but often, the larger it is, the more severe it is. On the other hand, the VCW, the narrowest portion of the regurgitant jet passing through the valve, is a more practical measurement (independent of technical factors, flow, and driving rate). The American Society for Echocardiography recommends VCW measurement in the parasternal long-axis (PLAX) view, focused on the MV with an imaging plane optimized for the VC with PFC and VC and MR jet aligned in the same plane. Mild MR will have a VCW of less than 0.3 cm, and severe MR will be seen with a VCW of ≥0.7 (Fig. 9b and c). Continuous wave Doppler through the regurgitant jet, while optimizing to align the insonation beam with the regurgitant flow, allows for assessing spectral waveform density and shape. The density of the CWD envelope of the regurgitant jet is proportional to the flow (i.e., the amount of red blood cells). Therefore, a faint or incomplete jet on CWD is compatible with mild MR, but the operator must keep in mind that the density is gain-dependent. Regarding shape, a triangular contour of the jet with an early MR peak velocity denotes a hemodynamically significant regurgitant jet; while not sensitive, it is compatible with severe MR compared to a more rounded parabolic envelope (Fig. 9d). While the above methods are simple to perform, attention to detail in Doppler angle, color, and Doppler gain and accurate measurement techniques remain crucial for accurately assessing MR severity.

3.5.4. Additional guidance

The abovementioned components should be sufficient to trigger the critical actions of comprehensive echocardiography and consultation with cardiology and cardiothoracic surgery. One other method that is more familiar to the advanced emergency point-of-care user is assessing the mitral valve inflow, as the E wave is often very high (120 cm/s and above). By using PWD in A4Ch view, with gate at the coaptation of MV leaflets, one can measure peak velocity; the thought being a large regurgitant volume causes a large rush of blood back into the LV creating large velocities. On the contrary, an A wave dominant pattern (A wave greater than E wave), is typically not supportive of severe mitral regurgitation [89].

Several other methods were not included, given their scope and complexity. Multiple quantitative methods are used in comprehensive echocardiography to navigate the severity of chronic MR; which are time-consuming, and minor errors can result in faulty measurements (effective regurgitant orifice area, the regurgitant volume/volume overload, and regurgitant fraction) [90,91]. Another method to check for severe MR includes assessing PWD through the pulmonary veins. If more than one has systolic flow reversal, this can be specific for severe MR (85–92 %) but can be challenging in acquisition and interpretation from a transthoracic approach [91,92].

3.5.5. Pitfalls

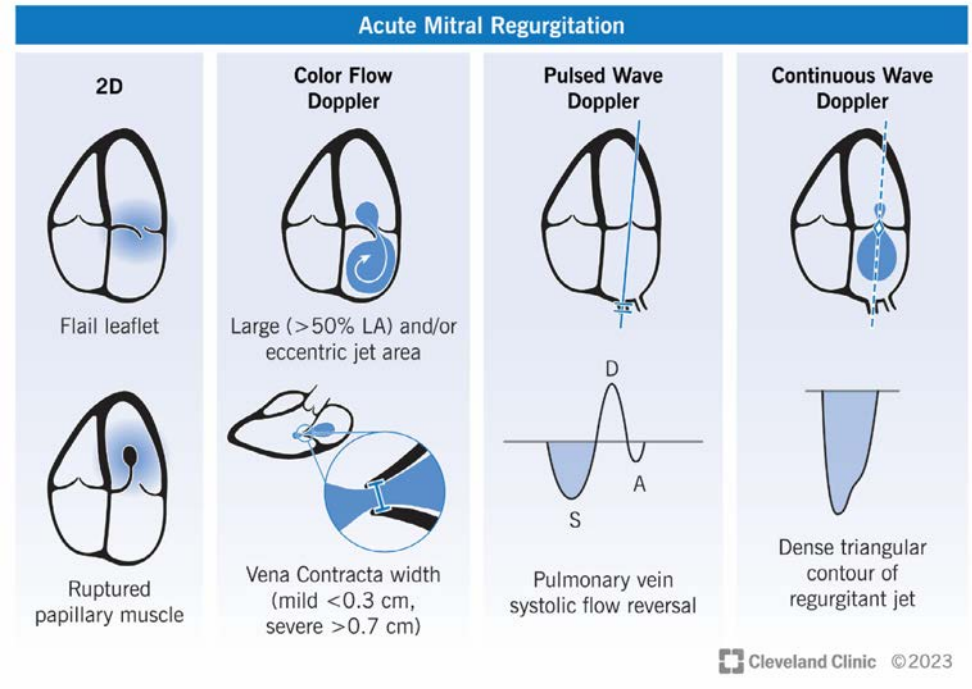
While each method of assessing MR has its advantages and disadvantages, the EP must integrate all the echocardiographic views and calculations along with the clinical variables to truly assess the etiology and severity of MR. The clinical situation may also increase the challenges of acquisition; for example, patients with cord or papillary rupture are often very tachycardic with challenging windows and likely suboptimal color flow.

It is not advisable to rely on a single view or calculation to assess MR. On 2D, one could easily miss subtle abnormalities in valve coaptation and excursion indicating present or impending flail leaflet or valve failure. While color flow is easy to perform, it relies on properly adjusting color parameters to accurately assess and it may over or underestimate the severity of MR, not to mention its dependent nature on cardiac hemodynamics. CWD also relies on properly adjusting machine parameters and is not sensitive for evaluating severe regurgitation. And as previously mentioned, quantitative methods, while more accurate for severity, are complex, time consuming and not practical for point-of-care providers in acute settings. Nonetheless, with these limitations and pitfalls mentioned early bedside echocardiography provides invaluable information in the diagnosis and management of patients with significant MR.

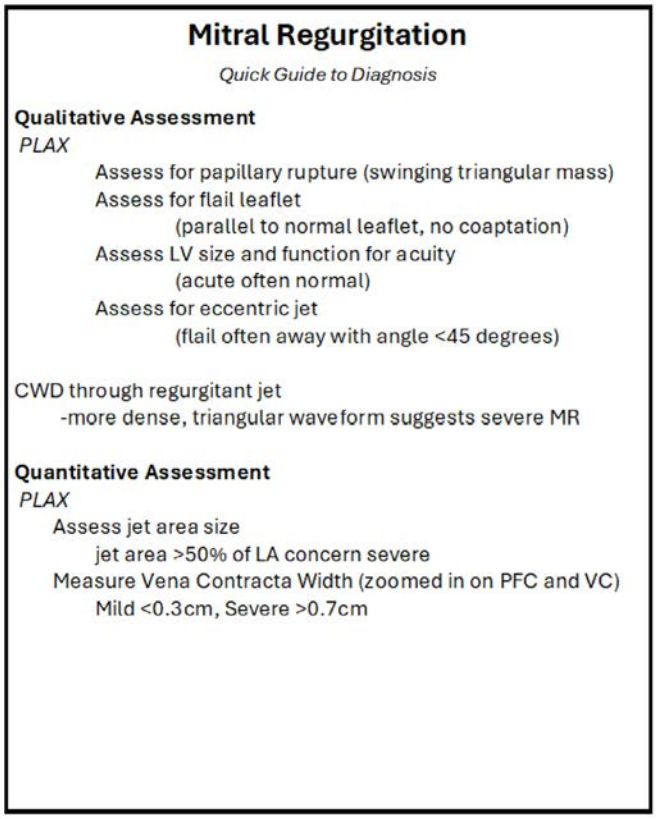
4. Conclusion

Focused echocardiograms are key to optimal diagnosis and management for critically ill patients in the ED. The focused questions generally should not change; however, the evaluations described above may increase the index of suspicion for or help identify a valvular emergency needing a different treatment, emergent cardiology echocardiogram, consultation, and/or different disposition. Various factors can influence the accuracy of point-of-care testing, including clinical environment, resource availability, image quality and complexity of ultrasound machines, provider skill, and comfort. In light of this, one should always consider a comprehensive, cardiology-performed echocardiogram in these scenarios to confirm point-of-care findings. Further studies are

A. Acute Mitral Regurgitation Illustrated Infographic



B. Acute Mitral Regurgitation Diagnosis Reference Card



Mitral Regurgitation

Quick Guide to Diagnosis

Qualitative Assessment

PLAX

Assess for papillary rupture (swinging triangular mass)
Assess for flail leaflet
(parallel to normal leaflet, no coaptation)
Assess LV size and function for acuity
(acute often normal)
Assess for eccentric jet
(flail often away with angle <45 degrees)

CWD through regurgitant jet
-more dense, triangular waveform suggests severe MR

Quantitative Assessment

PLAX

Assess jet area size
jet area >50% of LA concern severe
Measure Vena Contracta Width (zoomed in on PFC and VC)
Mild <0.3cm, Severe >0.7cm

Fig. 10. Acute mitral regurgitation.

needed to identify the most effective methods to diagnose acute valvular emergencies quickly and accurately in the ED.

While the evidence for POCUS diagnosis of acute valvular emergencies continues to grow, there are some practical pieces that can be added to a focused echocardiogram: In the setting of undifferentiated shock and/or respiratory failure, we would recommend assessing the valves visually in B-mode, qualitatively with color Doppler and adding quantitative evaluations as appropriate based on qualitative findings and the

clinical picture. Lastly, LVOTO assessment may be beneficial in patients with undifferentiated shock or shock not responding to usual resuscitative measures.

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CRediT authorship contribution statement

Robert Stenberg: Writing – review & editing, Visualization, Supervision, Project administration, Conceptualization. **John Bowling:** Writing – review & editing, Writing – original draft, Visualization, Investigation. **Joshua Jacquet:** Writing – original draft, Visualization, Investigation. **Kevin Watkins:** Writing – review & editing, Visualization, Conceptualization. **Justin Eggleston:** Writing – original draft, Conceptualization. **Andrew Hill:** Writing – original draft, Investigation. **Jessica Krizo:** Writing – review & editing, Supervision, Project administration, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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